manometer), pulmonary arterial pressure (PAP, Swan-Ganz catheter), and O_2 extraction ([arterial O_2 content – venous O_2 content]/arterial O_2 content). Exercise capacity was assessed by peak VO_2 uptake VO_2) during upright cardiopulmonary exercise test before and after 3 months of CR. *Result:* Peak VO_2 increased significantly after CR (1224 ± 271 to 1520 ± 380 ml/min., $p < 0.01$). The improvement in peak VO2 after CR was best correlated with the change in O_2 extraction during exercise before CR ($r = -0.59$, $p < 0.01$), followed by the change in EDP ($r = -0.54$, $p < 0.02$), but not with changes in heart rate, AoP, PAP during exercise nor LV ejection fraction at rest before CR. *Conclusion:* Exercise capacity improves to a greater extent by CR in patients with a smaller increase in O_2 extraction during exercise before CR. This suggests that an improvement in exercise capacity after ET is largely afforded by an improvement in the peripheral O_2 extraction capacity rather than the cardiac mechanism. Thus, O_2 extraction during exercise before CR may be a useful predictor for the improvement in exercise capacity after CR in patients after AMI.

989-108 Effects of warm up on hemodynamic responses to submaximal exercise after myocardial infarction

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We studied the influence of warm up on hemodynamic characteristics during exercise in 14 patients (pts) (aged 25–59 years) 6–20 weeks after uncomplicated myocardial infarction. Pts underwent invasive hemodynamics at baseline (R1) and during two similar consecutive submaximal rectangular supine bicycle exercise (E1 and E2) (25 Watts workload during 10 min) separated by a 10 min rest period (R2).

Results: 1/ Comparing R1 versus R2 hemodynamic parameters were not significantly different. 2/ Comparing E2 versus E1 heart rate, systolic and diastolic arterial pressures, cardiac index, systemic and pulmonary vascular resistances, oxygen consumption, arterial blood lactate concentrate, left ventricular stroke work were not significantly different whereas during E2 there is a significant decrease ($p < 0.05$) of mean pulmonary capillary wedge pressure (from 16.5 ± 10.5 to 9.7 ± 8.5 mm Hg), of mean pulmonary arterial pressure (from 24.8 ± 12.2 to 18.8 ± 10 mm Hg) and of right ventricular stroke work (from 16.3 ± 6.6 to 12.5 ± 7 gm.m).

Conclusions: Warm up exerts beneficial "trinitrine like" hemodynamic effects during submaximal exercise and improved exercise tolerance after myocardial infarction: it might be thus considered as an effective non pharmacological treatment.

989-109 Effects of Exercise Intensity on Short-Term Exercise Training in Patients With Acute Myocardial Infarction

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To examine whether the intensity of exercise training (TR) following acute myocardial infarction (AMI) influence the predischarge exercise response, 86 uncomplicated patients (pts) were studied. Seventy-four pts underwent upright bicycle TR 3 hr/week for 3 weeks. These pts were divided into 2 groups according to TR intensity: 70% of peak oxygen consumption (VO_2) (group A, n = 44), and 50% of peak VO_2 (group B, n = 30). Remaining 12 pts were restricted to minimal activity program as a control (group C). All the pts performed cardiopulmonary exercise testing before and after 3 week period. Before TR, there were no significant differences in exercise capacity, and hemodynamic and metabolic responses among the 3 groups. Heart rate, plasma norepinephrine levels, and arterial lactate concentration decreased significantly (all $p < 0.05$) at submaximal exercise after TR in group A (136 → 124 bpm, 1220 → 986 pg/ml, 40 → 36 mg/dl, respectively) and in group B (132 → 125 bpm, 1066 → 945 pg/ml, 37 → 33 mg/dl, respectively), but these indices were unchanged in group C. Peak cardiac output, stroke volume and VO_2 increased significantly (all $p < 0.001$) after TR in group A (9.1 → 9.9 L/min, 58.1 → 62.9 ml, 20.7 → 22.5 ml/min/kg, respectively), but these variables were unchanged after 3 weeks in groups B and C. *Conclusion:* Short-term TR at 50% of peak VO_2 after AMI improved sympathetic and metabolic responses to submaximal exercise, whereas TR at 70% of peak VO_2 improved exercise capacity and hemodynamic indices as well as sympathetic and metabolic responses to submaximal exercise.

989-110 Aerobic Capacity, Parasympathetic Modulation And Orthostatic Tolerance In Young Athletes

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We investigated the relationship between aerobic capacity, parasympathetic modulation, and orthostatic tolerance in 10 athletes. The subjects (N = 10; 7 male; 3 female) VO_2 maximum or maximal oxygen consumption ($ml \cdot kg^{-1} \cdot min^{-1}$) was determined by treadmill testing (low maximal oxygen consumption <40 $ml \cdot kg^{-1} \cdot min^{-1}$; moderate $>40-<55$ $ml \cdot kg^{-1} \cdot min^{-1}$; high ≥ 55 $ml \cdot kg^{-1} \cdot min^{-1}$). Frequency and time domain heart rate variability (HRV) analysis were used as indicators of parasympathetic modulation. Orthostatic tolerance was assessed by head upright tilt table (HUTT) testing. *Results:* Aerobic capacity groups; low; N = 2, moderate; N = 4, high; N = 4. The mean age was 25 ± 1 year. The frequency domain parameters; very low frequency (VLF), low frequency (LF), high frequency (HF), and the total of all frequency bands (TF) were significantly higher or had a greater rate of change in the high aerobic capacity group ($p < 0.05$, < 0.01, < 0.02, < 0.01), respectively. Time domain variables; the standard deviation about the mean of successive normal R-R intervals (SDNN), root mean square of the difference of successive R-R intervals (rMSSD), and percentage of successive normal R-R intervals greater than 50 msec (pNN50) were also significantly higher in the high aerobic capacity group ($p < 0.006$, < 0.02, < 0.05), respectively. A moderately-high correlation was determined to exist between time domain parameters and the HF component of frequency domain HRV analysis ($r \geq 0.82$). The correlation between VO_2 maximum and pNN50 was significant ($p = 0.82$). HUTT testing provoked two positive (20%) responses, one from the low and high aerobic groups. *Conclusions:* 1) Increased resting parasympathetic activity was a linear function of aerobic capacity. 2) Evidence exists for HRV analysis as a method for estimating aerobic capacity. 3) No association existed between aerobic capacity and the ability to withstand an orthostatic challenge. This information may be valuable in interpreting tilt test results in young athletic individuals.

989-111 The Protective Effect of Exercise on Acute Myocardial Infarction in Rat

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While regular exercise has been shown to prevent cardiovascular disease, few studies have shown that exercise is directly cardioprotective. We attempted to determine whether exercise prevents ischemic injury to the heart by using a rat model of myocardial infarction, produced by left coronary artery occlusion (20 min) followed by reperfusion (48 hr). One trial of exercise (Ex: running on treadmill for 30 min at 30 m/min, 0% grade) significantly reduced the size of infarct (IS) 0.5 and 48 hr after exercise in a biphasic manner compared with control group (C) without exercise. Pretreatment with N-2-mercaptopropionyl glycine, a scavenger of oxygen free radicals, during exercise (Ex+M) abolished the beneficial effect of exercise on infarct size both at 0.5 hr and 48 hr after exercise. When two trials of exercise were performed 48 hr apart (Ex2), the beneficial effect on infarct size strongly persisted for 0.5 to 72 hr after exercise. These results suggest that exercise directly protects against myocardial ischemia-reperfusion injury via a mechanism related to oxygen free radicals generated during exercise. Regular exercise may provide stronger protection against myocardial ischemia.

IS (%)	C	0.5 hr	24 hr	36 hr	48 hr	60 hr	72 hr
Ex	51 ± 2	21 ± 2*	48 ± 1	35 ± 2*	23 ± 2*	40 ± 2*	49 ± 2
Ex+M		51 ± 3			50 ± 2		
Ex2	51 ± 2	7 ± 1*	12 ± 2*	20 ± 2*	22 ± 2*	25 ± 3*	45 ± 2*

Mean ± SEM, *p < 0.05 vs C

989-112 Extreme Athletic Competition Does Not Induce Myocardial Injury

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In a recent ultramarathon, five participants were studied and all developed elevations in serum levels of cardiac troponin T (cTnT) suggestive of myocardial injury, using the Boehringer-Mannheim ELISA (first generation) assay. To determine if this elevation was due to actual myocardial injury or represented cross-reactivity of the cTnT assay with skeletal muscle TnT (smTnT), the current study was performed using a revised assay (Boehringer-Mannheim Enzymun) which exhibits minimal cross-reactivity between cTnT and smTnT. The Western States Endurance Run involves an arduous 100 mile continuous run through the wilderness of the Sierra Mountains, including severe

Table

(Normal)	CK (<150 μ/l)	CK-MB mass (< 5.0 ng/ml)	cTnT ELISA (< 0.2 ng/ml)	cTnT Enzymun (< 0.2 ng/ml)
Pre-race	0–314	0.7–9.0	0–0.01	0–0.09
Post-race	1905–69641	39–3056	0.01–9.23	0–0.16
Abnormal	23/23	23/23	21/23	0/23

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